Amendments to the Claims:

This listing of claims will replace all prior versions and listings of claims in the application.

Listing of Claims:

Claims 1-28 (Canceled)

29. (Currently amended) A transgenic mouse whose genome comprises a null ~~disruption in an~~ endogenous ~~murine~~ CX2 ~~gene~~allele~~, wherein where the disruption is homozygous, the transgenic mouse lacks production of functional CX2 protein, and exhibits, relative to a wild-type mouse, at least one of increased seizure susceptibility, increased glucose tolerance, and increased ability to metabolize glucose~~.

30. (Currently amended) The transgenic mouse of claim ~~29~~46, wherein the increased seizure susceptibility is characterized by a decreased response threshold to metrazol, relative to a wild-type control mouse.

31. (Currently amended) The transgenic mouse of claim ~~29~~46, wherein the increased glucose tolerance or increased ability to metabolize glucose is characterized by a decrease in blood glucose level after administration of glucose, relative to a wild-type mouse.

32. (Previously presented) A cell or tissue obtained from the transgenic mouse of claim 29.

33. (Canceled)

34. (Canceled)

35. (Canceled)

36. (Currently amended) A method of producing ~~a~~ the transgenic mouse of claim 1~~comprising a disruption in an endogenous murine CX2 gene~~, the method comprising:

    a. introducing a targeting construct capable of disrupting the endogenous murine CX2 gene into a murine embryonic stem cell;

    b. selecting for the murine embryonic stem cell which has undergone homologous recombination;

    c. introducing the murine embryonic stem cell selected for in step (b) into a mouse blastocyst;

    d. implanting the resulting blastocyst into a pseudopregnant mouse, wherein the resultant mouse gives birth to a chimeric mouse; and

    e. breeding the chimeric mouse to produce the transgenic mouse~~,~~

~~f. wherein where the disruption is homozygous, the transgenic mouse lacks production of functional CX2 protein and exhibits, relative to a wild-type mouse, at least one of increased seizure susceptibility, increased glucose tolerance, increased ability to metabolize glucose, increased body weight, increased body length and increased body weight to body length ratio.~~

37. (Canceled)

38. (Previously presented) A targeting construct comprising:

    a. a first polynucleotide sequence homologous to at least a first portion of an endogenous murine CX2 gene;

    b. a second polynucleotide sequence homologous to at least a second portion of the endogenous murine CX2 gene; and

    c. a selectable marker gene located between the first and second polynucleotide sequences.

39. (Previously presented) A method of producing a targeting construct, the method comprising:

    a. providing a first polynucleotide sequence homologous to at least a first portion of an endogenous murine CX2 gene;

    b. providing a second polynucleotide sequence homologous to at least a second portion of the endogenous murine CX2 gene;

    c. providing a selectable marker gene; and

    d. inserting the first sequence, second sequence, and selectable marker gene into a vector such that the selectable marker gene is located between the first and second sequences to produce the targeting construct.

40. (Currently amended) The transgenic mouse of claim ~~29~~ 43 wherein said mouse is a female mouse, said female mouse exhibits, relative to a wild-type control female mouse, at least one of increased body weight, increased body length and increased body weight to body length ratio.

41. (Canceled)

42. (New) The transgenic mouse of claim 29 wherein said mouse is heterozygous for said null allele.

43. (New) The transgenic mouse of claim 29 wherein said mouse is homozygous for said null allele.

44. (New) The transgenic mouse of claim 29 wherein said null allele comprises a gene encoding a selection marker.

45. (New) The transgenic mouse of claim 44 wherein said null allele further comprises a *lacZ* gene.

46. (New) The transgenic mouse of claim 43 wherein said mouse exhibits, relative to a wild-type control mouse, at least one of increased seizure susceptibility, increased glucose tolerance, and increased ability to metabolize glucose.